WHAT IS CLAIMED IS:

1. A method for screening a test compound for the ability to activate transcription through an indirect estrogen response, the method comprising:

a) providing a cell comprising an estrogen receptor and a promoter comprising an AP1 site which regulates expression of a reporter gene;

b) contacting the cell with the test compound; and

c) detecting the expression of the reporter gene.

2. A method of claim (a), wherein the cell is an Ishikawa cell.

3. A method of claim (a), wherein the cell over-expresses the estrogen receptor.

4. The method of claim (a), wherein the promoter is genetically engineered to comprise an AP1 site.

5. The method of claim (a), wherein the test compound is known to have antiestrogenic activity.

6. The method of claim (a), wherein the cell is derived from uterine tissue.

7. The method of claim 5, wherein the cell is a HeLa cell or an Ishikawa cell.

8. A method of claim (a), further comprising the steps of:

a) providing a second cell comprising an estrogen receptor and a promoter comprising a standard estrogen response element which regulates expression of a second reporter gene;

b) contacting the second cell with the test compound; and

c) detecting the expression of the second reporter gene.

9. A method of claim 7, wherein the response element is from the Xenopus vitellogenin A2 gene.

10. A method of claim (a), wherein the cell further comprises a promoter comprising a standard estrogen response element which regulates expression of second reporter gene.

11. A method of claim 9 wherein the response element is from the Xenopus vitellogenin A2 gene.

12. An estrogen agonist identified by the method of claim (a).

13. A method for screening a test compound for the ability to inhibit transcription through an indirect estrogen response, the method comprising:

a) providing a cell comprising an estrogen receptor and a promoter comprising an AP1 site which regulates expression of a reporter gene;

b) contacting the cell with the test compound and a compound known to mediate an indirect estrogen response;

c) detecting the expression of the reporter gene.

14. The method of claim 12, wherein the compound is known to mediate an indirect estrogen response is tamoxifen.

15. A method of claim 12, wherein the cell over-expresses the estrogen receptor.

16. The method of claim 12, wherein the promoter is genetically engineered to comprise an AP1 site.

17. A compound identified by the method of claim 12.

18. A method for screening a test environmental compound for estrogenic activity, the method comprising:

a) providing a cell comprising an estrogen receptor and a promoter comprising an estrogen response element which regulates expression of a reporter gene;

b) contacting the cell with the test compound; and

c) detecting the expression of the reporter gene.

19. The method of claim 17, wherein the cell further comprises a promoter comprising an AP1 site which regulates expression of a second reporter gene.

20. The method of claim 17, wherein the reporter gene is CAT.

21. The method of claim 17, wherein the cell over-expresses the estrogen receptor.

22. The method of claim 17, wherein the cell is an ERC1 cell.

23. A method of inhibiting agonistic activity of an antiestrogen compound, said method comprising administering with said antiestrogen compound an inhibitor selected from the group consisting of genistein, staurosporine, 6-thioguanine, and 2-aminopurine.

24. The method of claim 22, wherein said inhibiting agonistic activity comprises inhibiting an indirect estrogen response.

25. The method of claim 22, wherein said antiestrogen compound is tamoxifen.

26. The method of claim 22, wherein said inhibition is *in vivo*.

27. An estrogen response inhibiting composition comprising an inhibitor of a classical estrogen response pathway and an inhibitor of an indirect estrogen response.

28. The composition of claim 26, wherein said an inhibitor of a classical estrogen response pathway is tamoxifen.

29. The composition of claim 26, wherein said an inhibitor of an indirect estrogen response is selected from the group consisting of genistein, stauorsporine, 6-thioguanine, and 2-aminopurine.